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Repeated Assessment of Coronary Flow Velocity Pattern in Patients With First Acute Myocardial Infarction

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OBJECTIVES The aim of this study was to evaluate the coronary blood flow velocity pattern immediately and 24 h after percutaneous transluminal coronary angioplasty (PTCA) for acute myocardial infarction (AMI) in relation to myocardial reperfusion and follow-up left ventricular (LV) function.

BACKGROUND Analysis of coronary blood flow velocity pattern after AMI may provide information about microvascular damage and the occurrence of a reperfusion injury.

METHODS Measurement of coronary blood flow velocity pattern was performed immediately after PTCA and after 24 h in 25 patients with first AMI using a Doppler guidewire. Measurements were related to reperfusion determined by intravenous myocardial contrast echocardiography (MCE) performed before PTCA and at 24 h and to LV function at four weeks.

RESULTS Using MCE, 13 patients showed reperfusion and 12 patients showed nonreperfusion. Compared with patients with reperfusion, patients with MCE nonreperfusion had a lower systolic peak flow velocity immediately after PTCA (10.0 ± 0.3 cm/s vs. 19.3 ± 0.8 cm/s, respectively) and after 24 h (12.3 ± 0.4 cm/s vs. 21.3 ± 0.1 cm/s, respectively, $p = 0.0022$), more frequent early systolic retrograde flow (6/12 vs. 0/13, $p = 0.0052$ immediately after PTCA and 24 h later) and a shorter diastolic deceleration time immediately after PTCA (483 ± 6 ms vs. 737 ± 0 ms, respectively) and after 24 h (551 ± 9 ms vs. 823 ± 2 ms, respectively, $p = 0.0091$). Similarly, patients with impaired LV function at four weeks had altered coronary flow pattern compared with patients with preserved function. The coronary flow velocity pattern showed a tendency for improvement after 24 h in the reperfusion and the nonreperfusion groups.

CONCLUSIONS The coronary flow velocity pattern immediately and 24 h after PTCA for AMI relates to myocardial perfusion determined by MCE and LV function at four weeks. The flow velocity pattern shows slight improvement during the first 24 h after revascularization, indicating the absence of a major reperfusion injury. (J Am Coll Cardiol 2002;39:1283–9) © 2002 by the American College of Cardiology Foundation

Early patency of the infarct-related artery is the primary objective in the treatment of acute myocardial infarction (AMI). In addition, reperfusion on the myocardial tissue level has become a major objective (1–3). Myocardial contrast echocardiography (MCE) has demonstrated substantial nonreperfusion in a considerable number of patients even after revascularization procedures (1,2,4,5). Myocardial nonreperfusion is associated with significant changes in coronary blood flow patterns. The acute coronary blood flow pattern of patients with revascularized but nonreperfusion AMI has been demonstrated to be characterized by the appearance of early systolic retrograde flow, diminished systolic antegrade flow and rapid deceleration of diastolic flow (6–8). Experimental studies have suggested the presence of a reperfusion injury in addition to the ischemic insult resulting in further impairment of microvascular and myocellular function even after revascularization (9,10). However, the existence of injury caused by reperfusion is controversially discussed (11), in particular as there has been a lack of a method to evaluate the impact of the proposed reperfusion injury on microvascular function under clinical circumstances. Alterations in microvascular function are likely to be reflected by changes in coronary blood flow patterns. Thus, changes in coronary blood flow patterns might give insight into altered microvascular function, thereby allowing analysis of the proposed reperfusion injury.

This study sought to evaluate the coronary flow pattern immediately after percutaneous transluminal coronary angioplasty (PTCA) for AMI and at 24-h follow-up and relate the findings to reperfusion defined by intravenous MCE as well as left ventricular (LV) function at four weeks.

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Abbreviations and Acronyms

AMI = acute myocardial infarction
ECG = electrocardiographic/electrocardiogram
GWMI = global wall motion index
LV = left ventricle/ventricular
MCE = myocardial contrast echocardiography
PTCA = percutaneous transluminal coronary angioplasty
TIMI = Thrombolysis In Myocardial Infarction

Repeated analysis of coronary flow patterns was performed to allow improved understanding of changes in microvascular function occurring after reperfusion.

METHODS

Study population. Twenty-five consecutive patients with first AMI undergoing revascularization by primary PTCA within 6 h after onset of pain were included in the study. Inclusion criteria were typical anginal pain lasting >30 min and ST-segment elevation of >0.2 mV in at least two contiguous electrocardiographic (ECG) leads. The research protocol was approved by the local institutional ethics committees. All patients gave written informed consent to participate in the study.

Coronary angioplasty and coronary blood flow analysis. Successful PTCA including stent placement was performed in all patients. Flow in the infarct vessel after PTCA was graded using the Thrombolysis In Myocardial Infarction (TIMI) flow classification (12).

Within 5 min after completion of interventional recanalization, the guidewire was exchanged for a 0.014-in. intracoronary Doppler-tipped flow wire (12 MHz, FloWire, Cardiometrics Inc., Mountain View, California) to perform intracoronary flow measurements. Placing the tip just proximal to the site of recanalization ensured that the sampling volume corresponded to the site of occlusion. This allowed assessment of flow to the entire region at risk. The ECG and blood pressure were monitored continuously. Twenty-four hours after intervention, Doppler measurements were repeated within 1 h of the follow-up MCE. Doppler flow velocity spectra were analyzed off-line to determine the following parameters: peak and mean antegrade coronary flow velocity in systole and diastole and systolic and diastolic flow duration, the calculated ratio of mean diastolic to systolic flow velocity and the deceleration time of diastolic velocity. The rate of decline in flow velocity in diastole was calculated as the diastolic deceleration rate. A pathologic rapid retrograde flow in early systole was defined as follows: negative peak velocity ≥10 cm/s and duration ≥60 ms as previously described (6). All measurements were repeated three times, and the mean value was calculated.

MCE. Intravenous MCE was performed using Sonazoid (NC100100, Nycomed-Amersham, Oslo, Norway), a recently developed contrast agent consisting of a lipid stabilized suspension of perfluorobutane microbubbles with a mean diameter of 2.4 to 3.5 μm. Immediately before, 1 h and 24 h (within 1 h of follow-up intracoronary Doppler) after primary angioplasty, MCE was performed by administration of up to three injections of Sonazoid at a dose of 0.030 μl microbubbles/kg body weight followed by a 10-ml saline flush. Image acquisition was performed using harmonic intermittent imaging triggered to every heart beat at end-systole with a phased array transducer at a mean transmit frequency of 1.8 MHz and a mean receive frequency of 3.6 MHz (Sonos 2500 LE or Sonos 5500, Philips Ultrasound, Andover, Massachusetts). Echocardiography machine settings were used as described recently (5). All images were stored digitally on magneto optical disc and on S-VHS videotape for off-line analysis.

Image interpretation of echocardiograms. All ultrasound images were evaluated at a core lab in Pisa, Italy, by an independent reader blinded to any clinical information (P. V.) as described previously (5). In the two- and four-chamber view, the length of the endocardial border corresponding to the part of the myocardium with no or poor opacification was measured. The sum of the endocardial border length measurements in the two apical views defined the size of the perfusion defect as described by Ito et al. (1). The perfusion defect size divided by the total endocardial border length in the two apical views defined the relative perfusion defect size. The MCE perfusion defect size measured 24 h after intervention divided by the MCE perfusion defect size before PTCA, the ratio to the risk region, was used to define a myocardial reperfusion group (ratio < 0.5) and a nonreperfusion group (ratio ≥ 0.5).

Left ventricular wall motion at four weeks was analyzed according to the 16-segment model of the American Society of Echocardiography, and global and regional wall motion indexes were calculated (13). Patients were divided according to LV function at four weeks into a group with preserved LV function (global wall motion index [GWMI] < 1.5) and a group with depressed LV function (GWMI ≥ 1.5) (14).

Statistical analysis. Continuous variables are presented as mean ± SD and were compared using Student *t* test or Wilcoxon test, as appropriate. Dichotomous variables were compared using chi-square statistics or Fisher exact test. Pearson and Spearman correlation coefficients were calculated as adequate. A generalized linear model with respect to repeated measurements was fitted to the coronary flow velocity and hemodynamic variables to investigate time, group and interaction effects. The comparisons of immediately after intervention to 24 h after intervention and reperfusion to nonreperfusion as well as interaction between time-point and group were analyzed by F-test. All p values ≤0.05 were regarded as statistically significant. Because of the explorative capacity of the p values, no alpha-adjustment was made. All statistical analysis was performed using SAS V8.2 Software (SAS Institute Inc., Cary, North Carolina).

Table 1. Clinical, Angiographic Data and Functional Outcome of Patients Belonging to the Reperfusion Group (Reduction of Initial MCE Perfusion Defect ≥50% at 24 h versus Nonreperfusion Group (Reduction of MCE Perfusion Defect <50%)

	Reperfusion Group (n = 13)	Nonreperfusion Group (n = 12)	p Value
Age (yrs)	58.2 ± 11.4	67.2 ± 9.5	0.045
Men	7/13 (54%)	9/12 (75%)	0.411
Infarct location (anterior/inferior)	2/11	9/3	0.005
Perk creatine kinase (U/l)	1,140 ± 766	1,892 ± 1115	0.060
Time from onset of symptoms to PTCA (h)	4.3 ± 1.5	4.4 ± 1.4	0.937
TIMI 3 after PTCA	13/13 (100%)	7/12 (58%)	0.015
Abciximab	2/13 (15%)	5/12 (42%)	0.201
Visible collaterals	5/13 (38%)	1/12 (8%)	0.160
RWMI before PTCA	2.6 ± 0.5	2.8 ± 0.2	0.135
RWMI after 4 weeks	1.9 ± 0.5	2.5 ± 0.4	0.003
GWMI before PTCA	1.6 ± 0.2	1.9 ± 0.2	0.003
GWMI after 4 weeks	1.4 ± 0.3	1.8 ± 0.3	0.003

GWMI = global wall motion score index; MCE = myocardial contrast echocardiography; PTCA = percutaneous transluminal coronary angioplasty; RWMI = regional wall motion score index; TIMI = Thrombolysis In Myocardial Infarction.

RESULTS

Study cohort and procedural outcome. Baseline clinical, angiographic and functional outcome data are shown in Table 1. Depending on the MCE examination, 12 patients were assigned to the nonreperfusion group and 13 patients to the reperfusion group. The two groups did not differ with respect to arterial blood pressure and heart rate during the intracoronary flow velocity measurements. Similarly, hemodynamic conditions during Doppler flow measurements immediately after PTCA and at 24 h did not differ significantly (Table 2). Figures 1 and 2 show typical MCE images and coronary blood flow velocity patterns in patients from the reperfusion and the nonreperfusion groups, respectively.

Coronary flow velocity patterns related to myocardial reperfusion. There was a significant correlation between the mean systolic flow velocity in the infarct-related artery immediately after revascularization and the relative perfusion defect size determined by intravenous MCE directly after angioplasty ($r = -0.719$, $p < 0.001$) and after 24 h ($r = -0.753$, $p < 0.001$), respectively. There was no significant correlation between the time from onset of symptoms until reperfusion by PTCA and any of the Doppler flow parameters.

Peak and mean systolic velocity as well as the systolic flow duration were significantly lower in patients with MCE nonreperfusion than in those with MCE reperfusion both immediately after the revascularization procedure as well as after 24 h (Table 2). Six of the 12 patients with MCE nonreperfusion had an early systolic flow reversal, whereas none of the patients with MCE reperfusion had this flow abnormality. The same result was found 24 h later. The diastolic deceleration time was significantly shorter in patients with MCE nonreperfusion than in those with MCE reperfusion. This difference was also unchanged after 24 h. The diastolic-to-systolic peak velocity ratio was significantly higher in patients with MCE nonreperfusion than in those with MCE reperfusion. There was no evidence of an interaction between the time-point of measurement (immediately vs. 24 h after PTCA) and the reperfusion status (reperfusion vs. nonreperfusion) for all parameters but the systolic coronary flow duration (Table 2).

Table 2. Coronary Flow Velocity Data and Hemodynamics Immediately After Intervention and After 24 h of Patients Belonging to the Reperfusion Group (n = 13) (Reduction of Initial MCE Perfusion Defect ≥50% at 24 h) versus Nonreperfusion Group (n = 12) (Reduction of MCE Perfusion Defect <50%)

	Immediately After Intervention		24 h After Intervention		Reperfusion vs. Nonreperfusion (Group)	Immediately vs. 24 h (Time-point)	Interaction Time-point Group*
	Reperfusion	Nonreperfusion	Reperfusion	Nonreperfusion			
Heart rate (beats/min)	74 ± 17	78 ± 17	70 ± 10	76 ± 18	0.4393	0.3605	0.8033
Systolic BP (mm Hg)	122 ± 27	121 ± 22	128 ± 29	110 ± 16	0.2781	0.7088	0.0982
Diastolic BP (mm Hg)	72 ± 16	72 ± 13	71 ± 14	66 ± 7	0.5954	0.1993	0.2996
APV (cm/s)	20.3 ± 8.6	15.2 ± 6.0	22.8 ± 11.1	15.1 ± 6.3	0.0211	0.5775	0.5504
Systolic coronary flow							
PV (cm/s)	19.3 ± 8.8	10.0 ± 4.3	21.3 ± 12.1	12.3 ± 5.4	0.0022	0.1427	0.7547
MV (cm/s)	15.9 ± 7.4	6.7 ± 3.7	18.2 ± 11.1	9.5 ± 4.9	0.0013	0.1520	0.8884
FD (ms)	315.8 ± 90.2	214.6 ± 103.3	291.0 ± 19.8	268.1 ± 76.7	0.0304	0.4740	0.0292
No. of ESFR	0/13	6/12	0/13	6/12	*		
Diastolic coronary flow							
PV (cm/s)	34.1 ± 17.5	37.1 ± 19.2	35.0 ± 17.2	30.6 ± 15.2	0.8280	0.5563	0.2845
FD (ms)	482.5 ± 120.4	455.8 ± 70.0	558.0 ± 109.8	514.1 ± 138.1	0.4370	0.0312	0.9248
DDR (cm/s^2)	54.4 ± 41.3	93.83 ± 55.8	55.1 ± 41.1	73.1 ± 54.6	0.0705	0.4253	0.3725
DDT (ms)	736.5 ± 220.1	482.6 ± 255.6	823.4 ± 432.1	551.6 ± 279.4	0.0091	0.3403	0.9213
DSVR	1.7 ± 0.9	3.7 ± 2.0	1.6 ± 0.8	2.4 ± 1.6	0.0025	0.0677	0.0940

P values of repeated measures analysis of variance with immediately respectively 24 h after intervention as repeated and reperfusion respectively nonreperfusion as group factor.
*p = 0.0052 using Fisher exact test.

APV = average peak flow velocity; BP = blood pressure; DDR = diastolic deceleration rate; DDT = diastolic deceleration time; DSVR = diastolic-systolic velocity ratio; ESFR = patients with early systolic retrograde flow; FD = flow duration; MCE = myocardial contrast echocardiography; MV = mean flow velocity; PV = peak flow velocity.

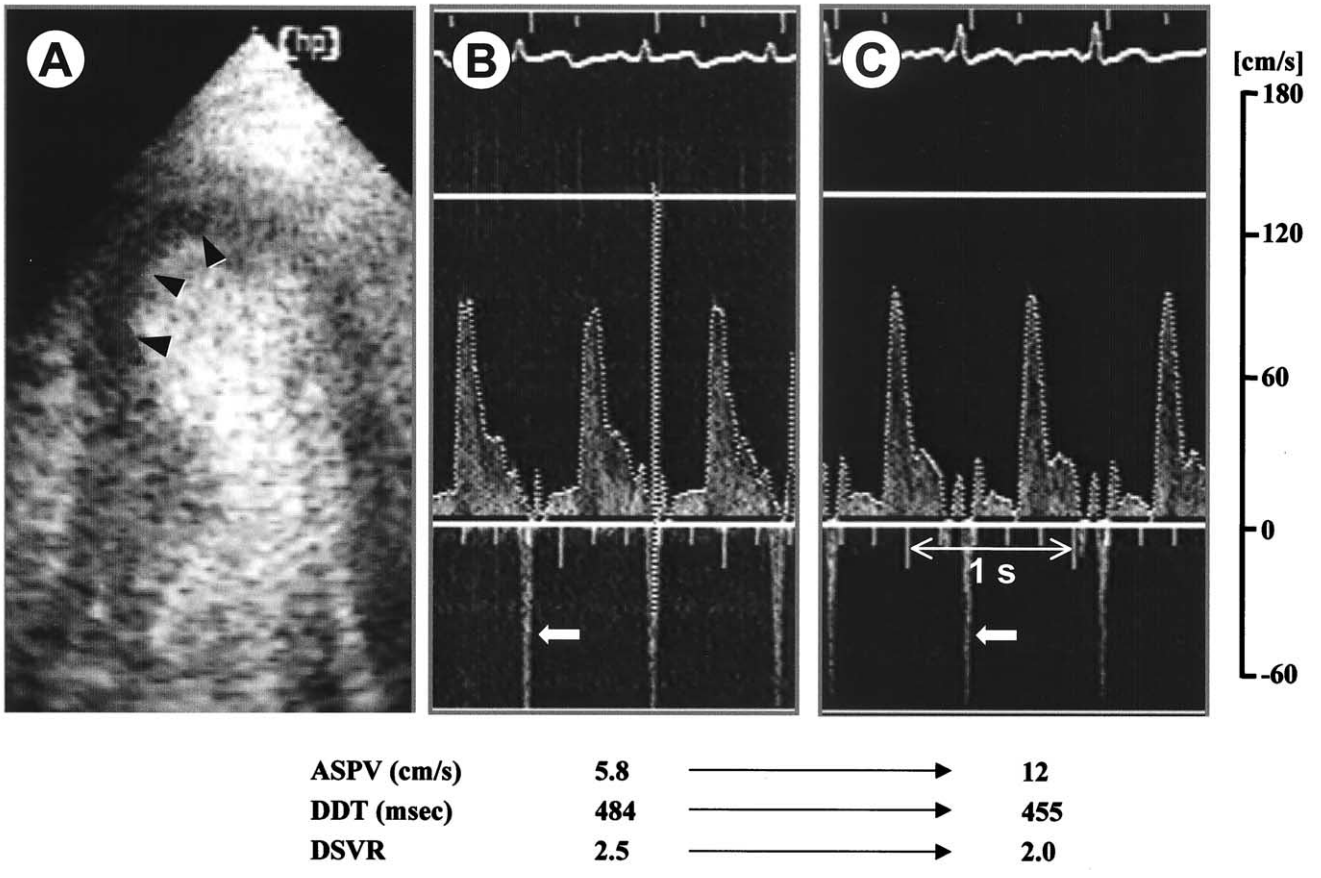

Figure 1. Myocardial contrast echocardiography (MCE) at end-systole (apical four-chamber view **[A]**) 24 h after percutaneous transluminal coronary angioplasty in a patient with nonreperfusion **(arrowheads)** defined by MCE after anterior acute myocardial infarction. The coronary flow velocity pattern in the left anterior descending artery immediately after revascularization **(B)** and at 24 h follow-up **(C)** showed retrograde systolic flow **(arrow)** and rapid deceleration of diastolic flow. There is a slight improvement in flow pattern during follow-up. ASPV = average peak flow velocity; DDT = diastolic deceleration time; DSVR = diastolic-systolic velocity ratio.

Coronary flow velocity pattern related to LV function. Average GWMI decreased from 1.67 ± 0.33 after revascularization to 1.59 ± 0.37 at four-week follow-up ($p < 0.05$). Fourteen patients had a depressed LV function (GWMI ≥1.5) at four weeks and 11 patients had a preserved LV function (GWMI <1.5). Compared with patients with preserved LV function, patients with depressed LV function at four-week follow-up had a lower peak systolic velocity immediately after PTCA (8.7 ± 5.5 cm/s vs. 15.1 ± 8.3 cm/s, $p < 0.05$), more frequent early systolic retrograde flow (6/14 vs. 0/11, $p < 0.05$, immediately after PTCA and 24 h later), a higher diastolic deceleration rate immediately after revascularization (95.1 ± 58.4 cm/s vs. 45.7 ± 22.6 cm/s^2, $p < 0.05$) and after 24 h (83.7 ± 52.0 cm/s^2 vs. 36.7 ± 24.2 cm/s^2, $p < 0.05$), a shorter diastolic deceleration time immediately after PTCA (520.5 ± 301.7 ms vs. 734.4 ± 155.4 ms, $p < 0.05$) and a greater diastolic-systolic velocity ratio immediately after intervention (3.4 ± 2.1 vs. 1.7 ± 0.6, $p < 0.05$) and after 24 h (2.3 ± 1.6 vs. 1.6 ± 0.7, $p < 0.05$).

Repeated assessment of coronary flow velocity pattern. All patients with an early systolic flow reversal immediately after revascularization showed this flow pattern also after 24 h. The peak systolic velocity increased by 22% from immediately after intervention to 24-h follow-up. Mean diastolic deceleration time increased slightly by 11% within the first 24 h after revascularization. The diastolic-systolic velocity ratio showed a trend towards improvement considering the time-point (Table 2). There was a similar trend for normalization of coronary flow velocity parameters between the reperfusion and nonreperfusion groups (Table 2).

DISCUSSION

This study demonstrates: 1) significant differences in coronary blood flow pattern immediately and 24 h after PTCA for AMI between patients with and without reperfusion defined by intravenous MCE; 2) significant differences in immediate coronary blood flow pattern between patients with preserved versus those with depressed LV function at four-week follow-up; and 3) slight improvement in coronary blood flow pattern 24 h after AMI.

Coronary flow pattern after AMI. There is a possible lack of reperfusion on the myocardial tissue level despite an open infarct-related vessel with TIMI 3 flow after revascularization for AMI (1,15). This "no-reflow" phenomenon is thought to be the result of microvascular dysfunction. It

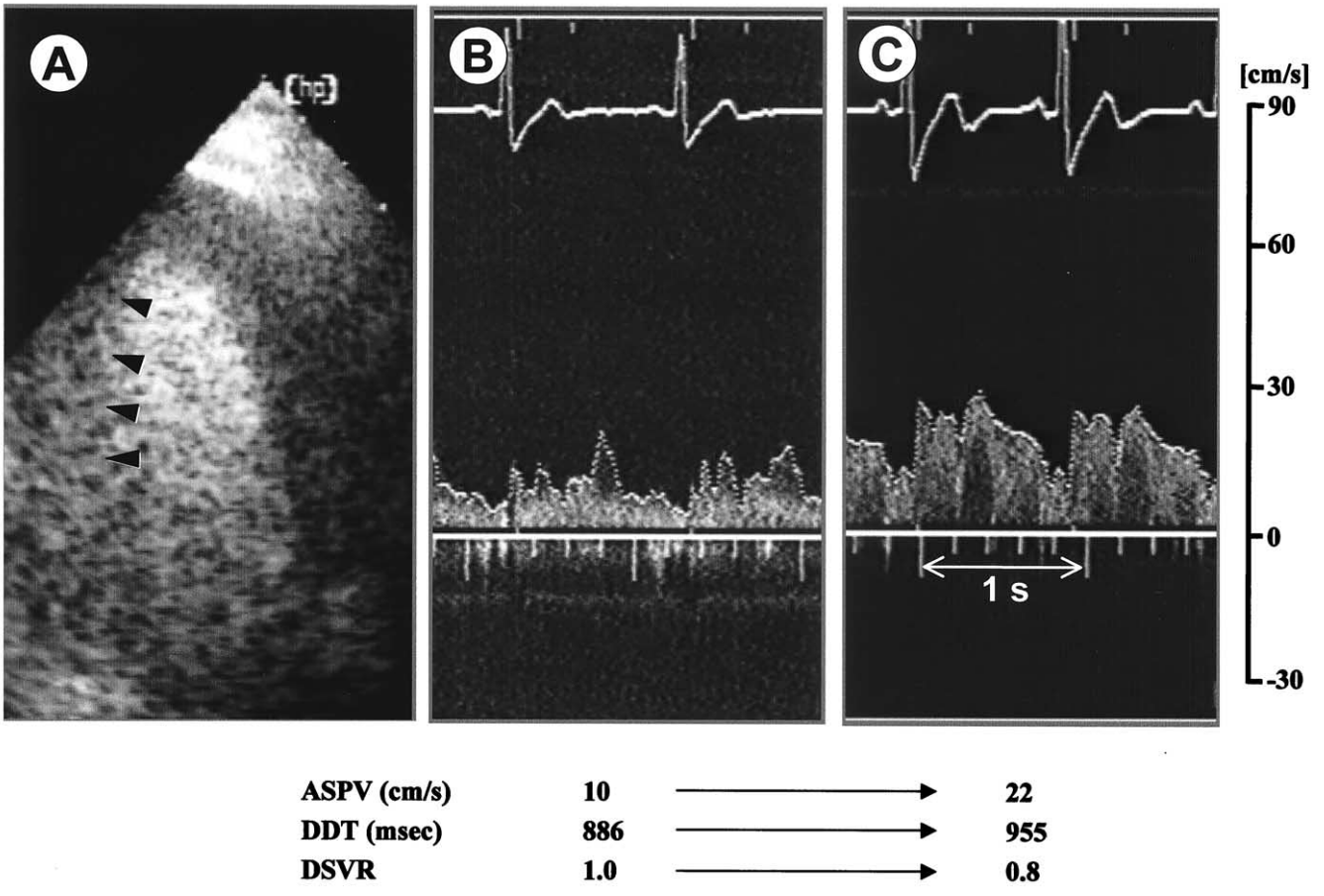

Figure 2. Myocardial contrast echocardiography (MCE) at end systole (apical two-chamber view **[A]**) 24 h after percutaneous transluminal coronary angioplasty in a patient with reperfusion **(arrowheads)** defined by MCE after inferior acute myocardial infarction. The coronary flow velocity pattern in the right coronary artery immediately after revascularization **(B)** and at 24-h follow-up **(C)** differs substantially from that seen in Figure 1. The coronary flow velocity pattern showed an increase in the average peak flow velocity (ASPV) from 10 cm/s immediately after revascularization to 22 cm/s at 24-h follow-up. DDT = diastolic deceleration time; DSVR = diastolic-systolic velocity ratio.

constitutes a marker of more extensive myocardial tissue damage and is associated with poorer functional recovery and patient outcome after AMI (2,4,5). Alterations in the coronary flow velocity pattern in patients with microvascular dysfunction after AMI have been demonstrated by Iwakura et al. (6,16). These alterations include impaired systolic antegrade flow, the appearance of abnormal retrograde flow in early systole and rapid deceleration of the diastolic flow velocity. An increase of microvascular impedance and a decrease of intramyocardial blood pool volume are known sequels of myocardial tissue damage. The increased microvascular impedance has been used to explain the impaired systolic antegrade flow as well as the systolic retrograde flow. The obstruction of the microvasculature with subsequent high impedance results in the inability to squeeze blood forward into the venous circulation during systole. It is consequently pushed back to the epicardial coronary artery to produce early systolic retrograde flow. The reduced intramyocardial blood pool, which fills rapidly during diastole, has been used to explain the rapid decline of diastolic velocity (6).

This study confirms recent reports on the difference in coronary blood flow pattern between patients with and without reperfusion determined by MCE (6–8). In contrast with recent reports that used intracoronary MCE to categorize patients into a reperfusion and a nonreperfusion group, intravenous MCE was applied in this study. Importantly, patients with significantly altered coronary flow pattern were found to have subsequent depression of LV function at four-weeks follow-up, confirming the prognostic importance of altered coronary blood flow patterns reported in previous studies (7,17).

Early changes in coronary flow pattern. While coronary Doppler flow studies allow assessment of microvascular circulation after AMI, serial Doppler studies may enable the determination of changes in microvascular function. In addition to the damage from the ischemic insult, a reperfusion injury has been discussed. Mechanisms such as calcium overload, mitochondrial injury, leucocyte accumulation and activation, osmotic stress and oxygen free radicals have been discussed as causes of a reperfusion injury. Several experimental studies have stressed an explosive release of oxygen free radicals after reperfusion of ischemic hearts and proposed the oxygen free radicals to be the primary cause of

reperfusion injuries (9,18). Vascular endothelial cells may demonstrate functional and morphologic abnormalities after exposure to oxygen free radicals. However, while experimental studies are indicative for the existence of reperfusion injuries, there is no conclusive evidence for a reperfusion injury under clinical conditions (19). Thus, the existence of a reperfusion injury has been controversially discussed (16,20,21). Part of the difficulty of evaluating the effect of the proposed reperfusion injury in clinical practice has been due to the lack of a means to accurately evaluate the magnitude of myocardial and microvascular damage. While crude clinical end points are insufficiently sensitive to differentiate the impact of myocardial ischemia from reperfusion injury, a means to reliably distinguish the influences of ischemia from reperfusion injury has not been established.

This study demonstrated a slight trend for improvement in the coronary flow pattern within 24 h after angioplasty for AMI. The small changes could be demonstrated for the systolic flow velocity, the systolic flow duration, the systolic-to-diastolic flow ratio as well as the diastolic deceleration rate. These changes indicate a consistent trend for improvement within all Doppler parameters used to determine microvascular dysfunction after AMI towards coronary flow velocity data reported for normal coronary arteries (22). This finding suggests a slight recovery of microvascular function from the ischemic insult during the first 24 h after AMI. As microvascular function reflects myocardial viability, the findings are indicative of the absence of an important reperfusion injury. Presence of a significant reperfusion injury should have resulted in further deterioration of the coronary flow velocity pattern. Most experimental studies indicating a reperfusion injury applied only a relatively short period of ischemia, with 90 min being a frequently applied period (9,10). In contrast, time of ischemia in this study was more than 4 h, reflecting ischemia times commonly observed in clinical practice. Thus, the findings of this study indicate the absence of an important reperfusion damage in a clinical setting as analyzed in this study. Any reperfusion injury is likely to be minor compared with the ischemic insult, confirming experimental studies of ischemic damage occurring even without reperfusion (23).

A slight improvement of coronary flow velocity pattern could be demonstrated for the reperfusion as well as the nonreperfusion group. Thus, there is a difference between the reperfusion and the nonreperfusion groups in the severity of the ischemic myocardial injury occurring until revascularization, which results in differences in the acute coronary flow velocity pattern. Once the initial insult has occurred, both groups show a tendency of recovery in microvascular function over a 24-h period.

Study limitations. This analysis included only a limited number of patients due to the complexity of the evaluation. The coronary flow velocity pattern may change with the position of the tip of the guidewire especially affecting serial analysis. However, in this study the stent could be used as a landmark with the tip of the Doppler guidewire being positioned just proximal to the stent. The normal flow in coronary arteries is characterized by a predominance of diastolic flow (22), a finding that is less marked in the right coronary artery. It is, therefore, possible that the differences in diastolic-systolic velocity ratio between the reperfusion and the nonreperfusion group might, in part, be attributed to the different distribution of the infarct-related artery in both groups. However, this does not relate to the other coronary flow pattern parameters. The elapsed time between complete reperfusion after PTCA and the measurement of the Doppler profiles was up to 5 min. Thus, a possible very early deterioration of coronary flow velocity patterns during the initial first minutes of reperfusion may have gone undetected.

Reperfusion injury may occur primarily on the microvascular level or on the myocyte level, and both levels may not necessarily be linked to each other. The method used in this study allows only assessment of a potential reperfusion-related injury on the microvascular level. It does not enable the analysis of reperfusion injuries on the myocyte level. It is unclear whether serial studies of coronary blood flow patterns are sufficiently sensitive to determine minor reperfusion injuries on the microvascular level. The absence of further deterioration in the coronary flow velocity pattern 24 h after revascularization indicates a stable microvascular function. However, this does not exclude ongoing injury on the myocellular level. There might be improvement of microvascular circulation occurring later than 24 h after myocardial infarction, which could not be demonstrated. However, due to the invasive nature of the applied method, we believed it to be inadequate to perform more than one follow-up study. Myocardial contrast echocardiography is still a rapidly evolving technique. The methodology of bolus intravenous MCE and machine settings used in this study relate to the best knowledge at the time of study initiation. A quantitative analysis of MCE may have yielded more accurate information on reperfusion defects. However, there is no generally accepted method for quantitative analysis of MCE, which accounts for both the size of the perfusion defect, as well as the degree of reperfusion deficit for any area at risk. The applied qualitative analysis refers to previously described use of MCE for definition of reperfusion defects (1,2,5).

Conclusions. Coronary flow velocity patterns relate to myocardial perfusion determined by intravenous MCE and LV function at four weeks. The coronary flow velocity pattern shows a slight improvement during the first 24 h after revascularization for AMI, indicating the absence of a major reperfusion injury. Recovery of microvascular function shows a tendency for recovery independent of the severity of the initial insult.

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